Amendments to the Specification:

Please amend the specification as follows:

Please replace paragraph starting at page 8, line 21, with the following rewritten paragraph:

In particular, the invention is directed to a mammalian RPTPκ protein or glycoprotein having the amino acid sequence of RPTPκ shown in FIG.3 (SEQ ID NO:1). In another embodiment is provided a functional derivative thereof. Preferably, the RPTPκ is of human origin, and has the amino acid sequence SEQ ID NO:2, as shown in ~~FIG. 15(1)-(3)~~ FIG. 15A-15E.

Please replace paragraph starting at page 8, line 28, with the following rewritten paragraph:

The invention is further directed to a nucleic acid molecule, preferably DNA, which may consist essentially of a nucleotide sequence encoding a mammalian RPTPκ having the nucleotide sequence (SEQ ID NO:3) ~~(FIG. 1(1)—1(5))~~(FIG. 1A-1H)). Preferably, the nucleic acid molecule consists essentially of a nucleotide sequence encoding human RPTPκ and having the nucleotide sequence (SEQ ID NO:4) or encodes a functional derivative thereof. The DNA molecule is preferably cDNA or genomic DNA. The invention is further directed to the DNA molecule in the form of an expression vehicle, as well as prokaryotic and eukaryotic hosts transformed or transfected with the DNA molecule.

Please add the following new paragraph at page 19, between lines 9 and 10:

FIG. 22E. Effect of a mutation of the furin cleavage site on the aggregation capabilities of cells containing expression vectors encoding an R-PTPκ cDNA. The aggregates formed by parental S2 strains were compared with the aggregates of an S2 strain transfected with expression vectors encoding an R-PTPκ cDNA in which the furin cleavage site had been mutated (CM) (Y.-P. Jiang *et al.* Mol. Cell. Biol. 13, 2942 (1993)), the aggregates of cells transfected with a cDNA encoding a catalytically inactive deletion mutant of R-PTPκ lacking most of the intracellular (PTPase) domain (Δ-PTP), and the aggregates of

cells transfected with a *wt* R-PTPκ cDNA (wt). The differences in aggregation intensity between the different forms of R-PTPκ may reflect protein expression levels. Mutation of the furin cleavage site left the vector adhesion behavior intact, suggesting that cleavage of the R-PTPκ proprotein is not required for induction of cellular aggregation. (Y.-P. Jiang *et al.* Mol. Cell. Biol. 13, 2942 (1993)). These numbers were provided by a Coulter-counter counting method, which counts the aggregates observed in a visual inspection. This method underestimates the aggregation levels because it only scores aggregates above a certain size threshold.

Please replace paragraph starting at page 91, line 19, with the following rewritten paragraph:

The observed aggregation correlating with expression of RPTPκ could be accounted for by either a homophilic binding mechanism, in which cell-cell binding is mediated by interaction between RPTPκ proteins on different cells within aggregates, or by binding of the RPTPκ proteins to a second cell-surface ligand intrinsic to the parental transfected cells. It was possible to distinguish between these two hypotheses by marking difference populations of cells with the fluorescent lipophilic dye 1,1'-dioctadecyl-3,3,3',3'tetramethylindocarbocyanine perchlorate (diI) (J. Schlessinger *et al.* Science 195, 307(1977)0, and then testing them for their ability to co-aggregate. In these experiments, RPTPκ expressing and non-expressing cells were labeled with diI, mixed with unlabeled cells of either RPTPκ expressing or non-expressing types, and the presence of cells of either type in the aggregates formed was monitored by fluorescence microscopy. The results are illustrated in ~~FIG. 23~~ FIG. 23A-23C. Strikingly, mixing of unlabeled, RPTPκ positive cells with labeled, RPTPκ negative cells led to the formation of aggregates consisting exclusively of unlabeled cells. Conversely, when the RPTPκ expressing cells were labeled and allowed to aggregate with unlabeled control cells, aggregates consisted entirely of labeled, demonstrating that diI labeling does not interfere with the aggregation capacity of the transfected cells. Mixing of labeled and unlabeled cells, both expressing RPTPκ, led to the formation of mixed aggregates consisting of cells of either staining type, thus confirming that both diI stained and unstained cells have the ability to coaggregate. These results suggest that

aggregation of the RPTPκ transfected cells requires the presence of the protein on all cells within the aggregate, implying a homophilic binding mechanism.